SOME PATHOLOGICAL AND CLINICAL ASPECTS OF ACQUIRED (FALSE) DIVERTICULA OF THE INTESTINE.

By Edwin Beer, M.D.,
of New York.

Only a few years ago acquired diverticula of the intestine were looked upon as pathological curiosities, but owing to the work of Klebs, Heschl, Hanau, Good, Hansemann, Fischer, and Graser, more attention has been paid to them, and gradually their importance in relation to other abdominal conditions is becoming known. Even at the present day the direct clinical bearing of this condition is difficult to estimate, as the collected facts are rather meagre.

According to the old classification, diverticula were divided into false and true. The former became identified with acquired, the latter with congenital. The distinction based on the anatomical construction of the walls of these diverticula lay at the bottom of the first classification, but now we know that this, in many instances, is no distinguishing feature. False diverticula were considered mucosal herniæ through the muscularis, their walls consisting of serosa, submucosa, and mucosa. True diverticula, on the other hand, were made up of all the layers of the bowel wall, and Meckel's diverticulum was practically the only representative of this class. Recent work has shown that diverticula similar in structure to the true may develop during the patient's lifetime, and, as they have nothing to do with an embryonic development or a congenital structure, they should not be called true diverticula.

Klebs, Edel, Sudsuki, and others have reported acquired diverticula not due to direct traction on the intestine, in which all the layers of the intestinal wall were represented. In addition, Neumann, Nauwerck, and Hansemann have reported some cases due to direct traction by tumors (myoma, angioma) or by an accessory pancreas. These need not concern us, as they are exceedingly rare. Their production is clearly due to traction, and if they give any clinical picture, it will be due either to the primary disease or to some such pathological change as makes the diverticulum of Meckel give rise to symptoms. Those diverticula of all the layers of the gut wall not due to traction are often called true diverticula, as they differ from the true in no essential detail, as far as their intrinsic structure is concerned. They differ, however, in that they are acquired, and in this respect they and the more common mucosal herniæ above mentioned are grouped together. Both are acquired and both often develop in the same intestine. At times diverticula, with all the layers of the intestinal wall, develop first, and in them the mucosa is pushed between the muscular bundles and a mucosal hernia results. From all this the distinction, based upon the time in which the diverticula develop, whether congenital or acquired, will surely appear simpler and lead to less confusion.

The acquired diverticula are found in the small intestine, in the appendix, in the colon, sigmoid flexure, and rectum. They are often multiple. At times hundreds have been found in one intestinal tract. It is fairly well agreed that they are most frequently found in the sigmoid, and that, in general, they are much more common than was once thought. Graser found diverticula ten times in twenty-eight cases, and in an investigation carried on by Sudsuki fifteen cases out of forty showed diverticula in the sigmoid. In the small intestine they are rather regular in their position with relation to the circumference of that part of the bowel. Here they develop at the mesenteric attachment, perhaps a little to one side of it, and by their growth gradually separate the leaves of the mesentery. Rarely do they become larger than a walnut—usually they are the size of a large pea or a chestnut; at times microscopic in size (Graser). In the large bowel no such regularity obtains and diverticula are found going into the mesentery or attached to some other part of the circumference of the bowel. Very often they develop in the haustra alongside one of the longitudinal muscular bands, and frequently they grow into the appendices epiploicæ.

As far as their causation is concerned, a unanimity of opinion does not exist. It is generally granted that old people alone show them, and especially such old people as have suffered from long-standing constipation. Klebs finds them in fat people, whereas Hansemann almost emphasizes the leanness of his subject. The former thought they were due to traction exercised on the bowel by the mesentery, whereas Hansemann favors the theory of pulsion from within the bowel. It is evident that Klebs' view would not explain those cases that had diverticula on the side opposite the attached mesentery—traction on the mesenteric side could hardly lead to the development of diverticula on the opposed side. Klebs pointed out that the diverticula on the mesenteric side were in close relation to the vessels of the bowel, and this has been verified by Hansemann and Fischer. On this point there is no doubt.

Heschl, Hanau, and Good, to find the cause of these diverticula, experimented by filling the intestines of human corpses with water and noting where the injected bowels ruptured. They were pleased to find that the rupture occurred regularly into the mesentery, and from this they concluded that here was the weakest part of the bowel; consequently just here diverticula developed. Hansemann went a step farther, verifying what had been "proven"—*i. e.*, the existence at the mesentery of a locus minoris resistentiæ, and, in addition, producing artificially false diverticula in the intestines of older people. These diverticula, he reports, ran along the sheaths of the veins of the intestine and were similar, anatomically, to those produced by nature during the life of the patients.

Even if we could accept all this work as directly applicable to living conditions, we would still have to explain those diverticula that do not grow into the mesentery. *Why should these be produced by other agents than those leading to the mesenteric diverticula? Why should not all old people have diverticula, provided their bowels have been constipated, for surely they all have similar venous supply?* These investigators have not explained the origin of these structures, and only the anatomical part of their work is reliable.

A priori, one might have supposed that intestines subjected to increasing internal pressure would rupture at the mesentery. Here the bloodvessels normally enter and the many holes or spaces between the muscle bundles are filled during life with tense vessels containing blood. In the dead intestine altogether different conditions obtain. These spaces are all empty and non-resistant to internal pressure, the whole mesenteric wall of the gut being more in the nature of a sieve than of normal intestinal wall. Moreover, the distention caused by the injection of a stream of water resembles but very little the distention of the bowel within the body. In a recent article by Philipowicz the difference between the distensibility of the normal and dead bowel is emphasized. He never succeeded in distending the bowel to nearly the size it had while distended in the body from volvulus. The bowel ruptured long before an equal distention was attained. Chlumsky's experiments on the intestines of dogs throw some light on this side of the question. When he injected small intestines that were still alive, not removed from the normal animal, the rupture occurred opposite the mesentery, whereas, as soon as he worked with intestines removed from the animal and dead some ten or more hours, they ruptured into the mesentery (Experiment 7). In his earlier experiments with conditions approximating the normal, the ruptures never took place into the mesentery (Experiments 1 to 6).

In our experiments the process is peracute, whereas in the life of the individual it is very slow and chronic. If these facts were not enough to invalidate the experiments that have been published, and which I have been able to verify in greater part, the fact that clinically such intramesenteric ruptures are most exceptional, if they ever occur, shows that this is not the weakest part of the gut during life, as the above investigators declared. Frequently, in cases of ileus, the peritoneum and underlying muscle layers tear and separate, not at the mesentery, but usually more or less opposite to its attachment.

Chlumsky has demonstrated the same in the thicker intestines of dogs. First the serosa tears, then the muscularis, and, interesting to say, the circular first, later the longitudinal, and last of all the strong submucosa. All this shows that the weakest portion of the gut wall is rather at some point on the side opposite the mesenteric attachment, rather than where Hanau, Heschl, Good, and others have placed it.

In studying this question, I proceeded as other investigators have done, and obtained similar results. The intestine, especially the small, ruptured almost regularly between the leaves of the mesentery, but now and again I obtained a rupture on the side opposite the mesentery. In the large bowel, in general, I had the same results, but here the ruptures were more often on the side where no mesentery was attached than in the small gut. The artificial diverticula reported by Hansemann I could not produce.

From all that has just been said, it will be seen that these experiments are valueless, because they do not show the weakest part of the living intestinal wall. Conclusions based on them, as to the origin of false diverticula, naturally must be invalid. Even if we granted that they were allowable, in trying to explain those diverticula that developed on the non-mesenteric side we would again be at a loss. But without going so far, it must be emphasized that the *supposed weakness at the mesentery does not exist, and, as it does not exist, it has nothing to do with the origin of acquired diverticula.*

In 1898 Graser added to our information on this subject, and tried, not without opposition, to connect chronic congestion of the bowel with the formation of the diverticula. A great deal of doubt has been thrown on this by the fact that congestion is not found in every case of diverticula. When the veins of the bowel are overfilled with blood, they must make more room for themselves and put aside the adjacent muscular fibres. This leads to the production of points of less resistance; in fact, almost reproduces the condition that we find when experimenting on the dead bowel. The enlarged spaces about the veins in which the blood is at a lower pressure, or the similar spaces more or less empty when the congestive condition is relieved temporarily by an increased arterial pressure, gives us a sieve-like condition of the mesenteric wall. Whereas Hansemann limited his application of the relation between veins and diverticula to mesenteric diverticula, Graser sees no necessity for such limitation and finds this relation between diverticula and veins, even in the non-mesenteric diverticula, as they occur in the sigmoid flexure. This view of Graser does not explain the diverticula that are not simple mucosæ hernial, but consist of all the layers of the gut wall; and the recent work of Sudsuki can be considered as a fairly conclusive overthrow of the congestion aspect of the question, though in some cases this may be a secondary factor in the development of diverticula. Not satisfied with disproving the general applicability of this theory, by demonstrating that only six out of his fifteen cases of acquired diverticula showed a condition of congestion and a cause for such a condition, Sudsuki, still clinging, more or less, to the older anatomical findings, propounded another theory. He regards a diminished resistance of the connective tissue about the veins of the bowel as the predisposing cause of this condition, but he also leaves the etiological factors of the non-mesenteric diverticula more or less in the dark, and does not by this theory give any adequate explanation of the diverticula with muscular layers in their walls (of which he reported six instances), which naturally cannot be due to changes in the connective tissue in the vascular spaces, for the whole wall does give way.

No one can deny, after studying acquired diverticula, that the veins and their sheaths have a definite influence on the development of diverticula on the mesenteric side of the intestine, but from the fact that these diverticula often develop on the non-mesenteric sides, where no large veins pierce the bowel wall, it is more than likely that those on the mesenteric side do no more than direct the path along which the diverticula develop. There must be a cause that underlies both the mesenteric and the non-mesenteric diverticula, both those that are made up of serosa, submucosa, and mucosa, and those with these plus the muscular layers. From the similarity of the anatomical picture, it is highly probable that one and the same cause is present, no matter where the diverticula develop. Simple pulsion, due to collections of gas and feces, is not sufficient. If these alone sufficed, all people so afflicted would have diverticula, which is not the case. There must be some change in the resistant powers of the gut wall. The fact that *diverticula occur in old people, in people whose intestines have been more or less worked out, as evidenced by constipation, points to a muscular deficiency, and in this muscular weakness*, I think, *the cause of the formation of false diverticula must be sought.* This diminished muscular strength will explain the production of diverticula in the mesenteric and non-mesenteric parts of the bowel, for in those patients where the repeated increases in internal pressure overcome the diminished muscular strength of the wall, diverticula must result, diverticula of the whole gut wall or of the mucosa, submucosa, and serosa. According to this view, the latter or common form would develop when the muscular weakness is localized to a small area of muscular tissue, and, as in the mesenteric diverticula, the mucosa would be most affected and pushed along the lines of least resistance. The weak muscle bundles would give and be pushed slightly to either side, as is seen in many early and very small diverticula. The numerous vessels on the mesenteric side, especially the veins, would probably point the further path, and the intimate relation to the veins, which obtains in mesenteric diverticula, would be developed. When larger areas of the wall have lost their muscular strength, naturally all the layers of the wall will be found in the diverticula. Moreover, those diverticula with only longitudinal layers of muscle cells in their walls are equally well explained by this view of the process (Chlumsky). It is very probable that the original view of Klebs is, in part, correct, especially with reference to the production of the diverticula in the small intestine, and that traction on the gut by the mesentery is a factor in the production of the necessary weakness at this point, where these diverticula so regularly occur in the small intestine. That this traction is a reality can be easily demonstrated by distending loops of intestine *in situ* or by noting the enormous tension of the mesentery in ileus cases. Naturally, in those parts of the intestine where no mesentery exists, this cannot be a factor.

The advantage of looking at this process in this way is manifold. It explains the production of all diverticula, no matter whether they are mesenteric or non-mesenteric in position, and no matter what their structure. It does not depreciate the changes that must develop in the muscular strength of an intestinal wall that has been overworked for a long time in emptying a bowel which is more or less in the position of a chronic ileus, due, perhaps, to torpidity of the rectum, without the ability to re-establish an equilibrium by adequate hypertrophy, not to mention the influence of old age. Moreover, it is in perfect consonance with experimental work and clinical observation.

So much for the causation of acquired intestinal diverticula. Before describing those changes in the diverticula which lead to serious consequences and give a more or less definite clinical picture, two facts must be emphasized. Diverticula in the large intestine, especially in the sigmoid flexure and rectum, are peculiar in that they alone are likely to undergo extensive pathological changes. Why diverticula in the small intestine are comparatively harmless cannot be definitely stated at the present moment. Judging, however, from the fact that in the large intestine hardened fecal masses which are lodged in the diverticula probably lead to the further changes, it can be said that the fluid consistency of the contents of the small intestine will probably account for the relative immunity of that part of the bowel from secondary pathological changes. In addition to this constant immunity of the small intestine, it must be emphasized that it is rather the exception than the rule for diverticula of the sigmoid and rectum to give rise to any symptoms. This probably explains the fact that these diverticula have received so little attention from clinicians. In the literature of this subject there are at least eighteen instances of inflammatory processes arising in these diverticula and leading to grave, often fatal, results.

If the mucous membrane of one of these diverticula in the sigmoid be examined, one can readily see an atrophic condition, in which both the epithelium and their glands are altered. Even before ulceration takes place there is visible an infiltration of round cells in the submucosa. As these diverticula are very often filled with hard fecal masses, ulceration of the mucosa, due perhaps to pressure, is liable to result. Such ulceration may lead to changes, both in the adjacent wall of the intestine, to similar inflammatory changes in the mesentery of the sigmoid, also to similar changes about the bowel. Moreover, should such an ulceration develop rapidly, a perforation into the free peritoneal cavity results. If, however, such an ulceration should lead to the formation of adhesions between the sigmoid and another hollow organ, the perforation will lead to an anastomosis between the intestine and adherent viscus. At times such a diverticulum ulcerates into the mesentery and an abscess is formed between its leaves. At other times such an abscess may form in the wall of the gut or about it, and this may perforate into a hollow viscus, thus establishing a connection between intestine and bladder, this being the organ usually involved by a fistulous tract.

When the inflammatory process about the diverticula in the wall of the intestine has led to marked connective-tissue growth, this, by its secondary cicatricial contraction, occludes the lumen of the bowel more or less completely. Rotter has brought out the fact that these stenoses of the sigmoid and rectum differ, very markedly, from stenoses due to other causes. These are peculiar, in the fact that they are produced not by cicatricial processes in the mucosa and submucosa, but by the chronic inflammatory process in the deeper layers of the gut wall. In addition to this form of stenosis, diverticula lead to stenosis, both as a result of inflammatory changes in the mesentery and perisigmoid adhesions. Both of these are usually combined with the first form described. Perhaps the ileus cases described by Riedel as due to sigmoid mesenteritis are, in some cases, the result of thickening in the mesentery and angular bending of the intestine, caused by primary changes in acquired diverticula.

How often the carcinomata of the sigmoid arise from the chronic ulcerations of one of these diverticula is hard to determine, because so little attention has been paid to these structures. That such a tumor can arise from the mucous membrane of such a diverticulum, a case reported by Hochenegg conclusively proves.

False diverticula in the appendix have been reported by Kelynak, Ribbert, Edel, Fischer, and Mertens. Whether the earlier cases are acquired diverticula is doubtful. In a recent article by Mertens, it has been definitely shown, however, that diverticula are found in this organ, and that, here also, they may prove of very serious consequence. He reports ulceration and perforation of one of these, corresponding with a similar case of perforation of the diverticulum in the sigmoid, which he also reported.

From this short sketch of the pathological conditions that have been found in the eighteen reported cases, it is evident that acquired diverticula are not always as harmless as we are wont to regard them.

As far as the clinical picture is concerned, it, perhaps, would be best to group the reported cases according to the pathological condition found at operation or post-mortem, rather than according to the diagnosis.

1. *Diverticula which Produced Stenosis of the Sigmoid or Upper Rectum.* Of these I have been able to find six instances in the literature. Graser, Fabris, Rotter, Koch, Waldvogel, and Georgi have reported these cases. All were not simple cases of stenosis; for instance, Georgi's case was complicated by an abscess; Waldvogel's by a fistulous tract between the sigmoid and the bladder. Rotter's case shows, in addition to the stenosis, an abscess in the mesentery of the sigmoid, which communicated with the lumen of the intestine through six perforated diverticula. Koch reported a case of Rotter, which also showed perisigmoiditis, with a fistulous tract running to the bladder wall. All these cases suggested the diagnosis of carcinoma of the sigmoid (Rotter's, of the cæcum) leading to intestinal obstruction. The exact condition of affairs was not evident until the involved intestine was examined in detail; then the condition which had seemed, both clinically and at the autopsy, and *in vivo* during the operation, malignant, proved to be nothing but an inflammatory change arising from an acquired diverticulum.

From this it is evident that the prognosis of chronic ileus, due to sigmoid stenosis, even in the presence of a tumor (*e. g.*, Koch's case, where a movable mass was felt in the left iliac fossa; Georgi's case, where a nodular, sharply defined, fixed mass occupied the left iliac fossa; Rotter's case, with a mass as big as a child's head, etc.) must be very guarded. A colostomy above such a stenosis naturally would relieve the patient of the obstructive symptoms, and, perhaps, some of the good results in cases of supposed carcinoma of the sigmoid that have lived for years after the colostomy are just such cases as the above.

2. *Diverticula which Lead to Perforation into the Peritoneum.* In 1870 Loomis reported a case of peritonitis due to infection from a false diverticulum, which, however, showed no sign of rupture. In Georgi's article three other cases are mentioned, two reported by Fiedler in 1868, and one by Hilbert; all three cases had acute perforative peritonitis. Mertens reports a fourth case of peritonitis due to acquired diverticula, which had perforated into the free abdominal cavity. In this case a man, aged forty-five years, who had been constipated on and off, suddenly, after straining, developed a perforative peritonitis. At the operation the origin of the peritonitis was not found, and the patient died.

These cases teach a very important lesson. The sigmoid must be examined in cases of perforative peritonitis when the surgeon is in doubt as to the source of infection.

3. *Diverticula that Lead to Abscesses or Localized Peritonitis in the Left Iliac Fossa.* Georgi's case is an excellent instance of diverticula leading to suppurative processes in the left iliac fossa. Also in his case, which was complicated (as said before under 1) by stenosis, the probable diagnosis of carcinoma was made. This patient died of peritonitis following an exploratory laparotomy, at which the

tumor was declared inoperable. Post-mortem examination showed that the peritonitis owed its origin to the rupture of a suppurative focus about the sigmoid flexure and in its wall. That the tumor, instead of being malignant, was purely inflammatory, due to acquired diverticula. Gussenbauer, according to Mertens, had, many years ago, a similar supposed case of carcinoma of the sigmoid. The local peritonitis, in fact the whole tumor, when the resected gut was examined proved to be inflammatory, due to changes in acquired diverticula.

Such cases make it very clear that we must think of false diverticula when we are brought face to face with inflammatory conditions in the left iliac fossa in elderly people.

4. *Diverticula Leading to Perforation into the Urinary Bladder or Densely Adherent to the Bladder.* These and the stenotic cases are perhaps the most important from a prognostic standpoint. Abnormal communication between the bowel and the bladder suggests usually the gravest of diseases and an inoperable condition. Most of these cases that are due to carcinomata probably never get into the literature, so that a statistical table, such as Chavannaz has published, is of doubtful value. Most surgeons and pathologists, undoubtedly, see such communications most commonly after carcinoma of the bowel. Mikulicz speaks of such as if that were his experience. Still, the fact that in twenty such cases of communication between the bowel and bladder Chavannaz found that nine were inflammatory in origin, five tubercular, four carcinomatous, and two traumatic, is of great interest to us in connection with the question of diverticula. How many of the inflammatory cases were due to perforation of these into the bladder is not known, though the following cases will show that this has repeatedly happened, and that such a possibility must be reckoned with in these bowel-bladder fistulæ.

In the literature I have been able to find several cases of perforation into the bladder due to acquired diverticula. In 1858 Sidney Jones, in the *Transactions of the London Pathological Society*, reports the case of a man who passed feces and gas in his urine, and, finally, died, as the result of difficulty in urinating, because of a fecal concretion in the bladder. At the post-mortem examination a free communication was found between the bladder and a perforated sigmoid diverticulum. According to Waldvogel, Czerny had a somewhat similar case. Waldvogel himself saw a case in which the patient, who had been passing gas in his urine, had a communication between the bowel and bladder by a sinuous fistula. From the fact that the gas was odorless and contained no sulphur, he concluded that it was produced in the bladder. He was able to isolate a gas-producing bacterium, which he thinks wandered in from the intestine by the fistulous tract. In Mertens' article another case attributed to Treves is reported. Koch's case, of which mention has already been made, under stenosis, showed intimate adhesions to the bladder wall, so that part of this had to be resected. This patient had urinary symptoms, and the natural suspicion before operation was primary carcinoma of the gut, with secondary involvement of the bladder.

Hepner recently reported another case in an old woman. The symptoms came on suddenly after lifting a heavy weight. She had fever and pain in the abdomen and vomiting. Gradually the urine became turbid, and before long she showed all the typical signs of an anastomosis between the bladder and sigmoid. The fistulous tract was tied off by operation, and the condition was clearly shown to be a sigmoid diverticulum which had opened into the bladder.

5. *Diverticula and Mesenteritis.* Most of the cases mentioned have shown more or less marked thickening and contraction of the mesentery. As said before, it still remains to be proven that such changes due to acquired diverticula lead to that form of chronic ileus which Riedel cures by non-operative means. Theoretically, this relation seems plausible, and in a recent article by Brehm this author tries to establish a definite relation between inflammation in diverticula, mesenteritis, and volvulus. In eleven out of twenty cases of volvulus he found well-marked mesenteric changes, but he did not prove the first link in the chain. Perhaps in time that will be made clear.

6. *Diverticula and Appendicitis.* Mertens has given most time to the study of this relation. He found, in 106 appendices taken from corpses, two instances of diverticula, and in twenty-eight diseased appendices removed by operation he found one diverticulum which had perforated during the attack. As the appendix itself is really nothing but a diverticulum and subject so frequently to disease, it is a little doubtful whether false diverticula in this location are of much moment in bringing on the trouble. Prognostically, of course, they are of little or no importance, in comparison with those cases in and about the sigmoid that simulate malignant growths.

7. *Diverticula and Carcinoma.* This relation is shown by just one case, that of Hochenegg (Thirty-first Congress of German Surgeons). The future alone can show how important in the production of carcinomata the chronic ulcerations in false diverticula are.

Whether such cases of intra-abdominal disease as I have just enumerated, due to acquired intestinal diverticula, will be recognized more frequently in years to come, it is difficult to say. Only twenty years ago Virchow spoke of acquired diverticula as an unusual pathological condition, but during these twenty years our views have changed. Perhaps the same will occur in the study of the intra-abdominal conditions due to false diverticula, and before long many cases like the eighteen here gathered will be reported. Then will we be in a better position, I trust, to differentiate between the inflammatory processes due to intestinal diverticula and those malignant diseases for which they are mistaken. Already we are in a position to profit from the reported cases, both in our treatment and in our prognosis.

LITERATURE.

Sudsuki. Ueber Divertikel am S. Romanum, Langenbeck's Arch., 1900, p. 708.
Hansemann. Ueber die Entstehung falscher Darmdivertikel, Virch. Arch., 1896, Bd. cxliv.
Fischer. False Diverticula of the Intestines. Journal of Experimental Medicine, 1900.
Klebs. Path. Anatomie, 1869, p. 271.
Hanau. Falsche Darmdivertikel, Virch. Arch., 1896, Bd. cxlv.
Edel. Ueber Erworbene Darmdivertikel, Virch. Arch., 1894, Bd. cxxxviii.
Waldvogel. Fistel zwischen Flexura sigmoidea u. Blase, etc., Deutsche med. Woch., 1902, p. 32.
Chlumsky. Ueber verschiedene Methoden d. Darmvereinigung, Beiträge z. klin. Chirurgie, 1899, Bd. xxv.
Rotter. Ueber Entzündliche Stricturen d. Colon, etc., Langenb. Arch., 1900, Bd. lxi.
Graser. Die Falsche Darmdivertikel, Langenbeck's Archiv, 1899, Bd. lix.
Graser. Ueber multiple falsche Darmdivertikel in der Flexura sigmoidea, Münchener med. Wochenschrift, May 30, 1899.
Mertens. Falsche Divertikel d. Flexura sigmoidea u. d. Processus vermiformis, Mettheilungen u. d. Grenzgebiet d. Med. u. Chir., Bd. ix.
Brehm. Ueber die Mesenterialschrumpfung in ihre Beziehung z. Volvulus der Flexura sigmoidea, Langenbeck's Arch., 1903, Bd. lxx.
Philipowicz. Mittheilungen u. inneren Darmverschluss, etc., ibid.
Mikulicz. Handb. d. prakt. Chir., 1900.
Koch. Ueber einfach entzündliche Stricturen d. Dickdarms, Langenbeck's Arch., 1903, Bd. lxx.
Fabris. Sui diverticoli acquisiti dell' intestino crasso, ref. Centbl. f. Chir., 1902, p. 1005.
Loomis. Peritonitis Due to False Diverticula, etc., New York Medical Record, 1870, vol. iv.
S. Jones. Transactions of the London Pathological Society, 1858.
Georgi. Ueber die Erwerbene Darmdivertikel u. seine praktische Bedeutung, Deutsche Zeitschrift f. Chirurgie, 1903, Bd. lxvii.
Hepner. Operativ geheilter Fall von Blasen-Dickdarmfistel, Beiträge z. klin. Chirurgie, 1903, Bd. xxxviii.

THE COMPLICATIONS OF AMŒBIC AND SPECIFIC DYSENTERY, AS OBSERVED AT AUTOPSY.[1]

AN ANALYSIS OF ONE HUNDRED AND TWENTY CASES.

BY CHARLES F. CRAIG, M.D.,
FIRST LIEUT. AND ASST. SURGEON, U. S. ARMY, PATHOLOGIST AND BACTERIOLOGIST TO THE U. S. ARMY GENERAL HOSPITAL, PRESIDIO OF SAN FRANCISCO, CALIFORNIA.

IN the study of post-mortems on patients dying from dysentery at the General Hospital, I have been impressed with the frequent occurrence of grave complications in this disease, and have paid special attention to the pathological conditions present in the various organs. These conditions have been so constant and so important that it has seemed to me that they should be considered more at length than they are now in most of the text-books upon medicine. In looking up the subject of the complications of dysentery, in the most recent works, including even those of the larger

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[1] With permission of the Surgeon-General of the United States Army. Read at the February, 1904, meeting of the San Francisco County Medical Society.